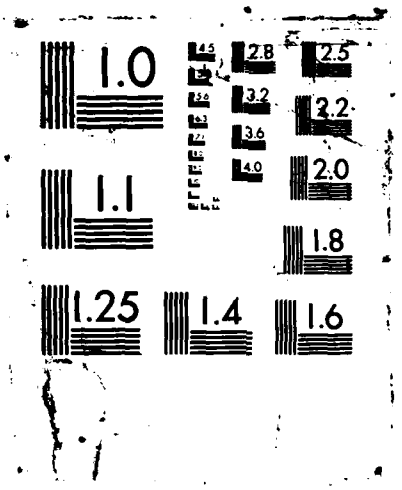
AD-A192 602 HYPERTHERMIA NEW THOUGHTS ON AN OLD PROBLEM(U) ARMY 1/1
RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA
R W HUBBARD ET AL. DEC 87 USARIEM-M-13/88

UNCLASSIFIED F/G 6/5 NL


1.0
1.1
1.25
1.4
1.6
1.8
2.0
2.2
2.5
2.8
3.2
3.6
4.0
4.5

UNCLAS
SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188
Exp. Date: Jun 30, 1986

1a. REPORT SECURITY CLASSIFICATION UNCLAS	1b. RESTRICTIVE MARKINGS
2a. SECURITY CLASSIFICATION AUTHORITY	3. DISTRIBUTION / AVAILABILITY OF REPORT DISTRIBUTION A: DISTRIBUTION STATEMENT A Approved for public release;
2b. DECLASSIFICATION / DOWNGRADING SCHEDULE	Distribution Unlimited
4. PERFORMING ORGANIZATION REPORT NUMBER(S)	5. MONITORING ORGANIZATION REPORT NUMBER(S)
6a. NAME OF PERFORMING ORGANIZATION US Army Rsch Inst of Env Med Heat Research Division	6b. OFFICE SYMBOL (If applicable) SGRD-UE-HR
6c. ADDRESS (City, State, and ZIP Code) Natick, MA 01760-5007	7a. NAME OF MONITORING ORGANIZATION 7b. ADDRESS (City, State, and ZIP Code)
8a. NAME OF FUNDING / SPONSORING ORGANIZATION	8b. OFFICE SYMBOL (If applicable)
8c. ADDRESS (City, State, and ZIP Code)	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER

10. SOURCE OF FUNDING NUMBERS

PROGRAM ELEMENT NO.	PROJECT NO.	TASK NO.	WORK UNIT ACCESSION NO.
61102A	3M161102BS15	CA	DA311249

11. TITLE (Include Security Classification)
Hyperthermia: New Thoughts On An Old Problem

12. PERSONAL AUTHOR(S)
Roger W. Hubbard, Ph.D., and Lawrence E. Armstrong, Ph.D

13a. TYPE OF REPORT	13b. TIME COVERED	14. DATE OF REPORT (Year, Month, Day)	15. PAGE COUNT
Manuscript	FROM _____ TO _____	December 1987	17

16. SUPPLEMENTARY NOTATION

17. COSATI CODES			18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
FIELD	GROUP	SUB-GROUP	Heat syncope, heat exhaustion, heat stroke, Energy Depletion Model, The Sodium Pump

19. ABSTRACT (Continue on reverse if necessary and identify by block number)
In this article, the common heat illnesses (heat syncope, salt-and water depletion-heat exhaustion and exertion-induced heatstroke) are briefly reviewed from the perspective of their underlying physiological disturbances, which provide a rational basis for therapy. Due to an apparent paradox between the current clinical dogma and laboratory data, however, the evaluation of the consequences of hyperthermia is extended beyond the usual systemic approach. This raises two new questions: what translates heat stress into heat strain at the level of the cell, and is a mechanism identifiable? A new hypothesis, based upon experimental research, is offered which suggests that a series of factors operate in exercise-induced hyperthermia to increase the permeability of the cell membrane, primarily to sodium ions. This stimulates sodium-potassium ATPase (the Sodium Pump) and results in an inefficient energy drain upon the cell (the Energy Depletion Model). This concept takes the form of a viscious circle leading to increased heat production and storage, reduced exercise-heat tolerance and significant morbidity and mortality. This model predicts that cellular/ metabolic processes and deficits operate for some time after hyperthermia has subsided w/cooling

20. DISTRIBUTION / AVAILABILITY OF ABSTRACT	21. ABSTRACT SECURITY CLASSIFICATION
☐ UNCLASSIFIED/UNLIMITED ☐ SAME AS RPT. ☐ DTIC USERS	UNCLAS

22a. NAME OF RESPONSIBLE INDIVIDUAL	22b TELEPHONE (Include Area Code)	22c OFFICE SYMBOL
Dr. Roger W. Hubbard	617-651-5153	

DD FORM 1473, 84 MAR 83 APR edition may be used until exhausted
All other editions are obsolete.

SECURITY CLASSIFICATION OF THIS PAGE
UNCLAS

Hyperthermia: New Thoughts On An Old Problem

Roger W. Hubbard, Ph.D

Lawrence E. Armstrong, Ph.D

LABORATORY: U.S. Army Research Institute
of Environmental Medicine,
Heat Research Division
Natick, MA 01760-5007
(617) 651-4871/4873

In brief

In this article, the common heat illnesses (heat syncope, salt-and water depletion-heat exhaustion and exertion-induced heatstroke) are briefly reviewed from the perspective of their underlying physiological disturbances, which provide a rational basis for therapy. Due to an apparent paradox between the current clinical dogma and laboratory data, however, the evaluation of the consequences of hyperthermia is extended beyond the usual systemic approach. This raises two new questions: what translates heat stress into heat strain at the level of the cell, and is a mechanism identifiable? A new hypothesis, based upon experimental research, is offered which suggests that a series of factors operate in exercise-induced hyperthermia to increase the permeability of the cell membrane, primarily to sodium ions. This stimulates sodium-potassium ATPase (the Sodium Pump) and results in an inefficient energy drain upon the cell (the Energy Depletion Model). This concept takes the form of a viscious circle leading to increased heat production and storage, reduced exercise-heat tolerance and significant morbidity and mortality. This model predicts that cellular/metabolic processes and deficits operate for some time after hyperthermia has subsided with cooling.

Introduction

As reviewed recently by Rowell (1), the potential conductance of the skin (7 $L \cdot min^{-1}$), splanchnic (3 $L \cdot min^{-1}$), and muscle (65-70 $L \cdot min^{-1}$) vasculature is enormous and far exceeds the pumping capacity (~22 $L \cdot min^{-1}$) of the normal human heart. Since the combined blood flow requirements of these vascular beds cannot be met during heat stress and upright exercise, there is an inherent competition between the mechanisms which maintain blood pressure and those which maintain blood flow to support muscle metabolism and thermoregulation.

The regulation of these competing demands is complex. For example, a rise in skin blood flow increases cutaneous venous volume, which not only enhances heat loss from the skin but also creates a reduction in effective central blood volume. It has long been held that a fall in blood pressure under these conditions is avoided due to a reduction in splanchnic and renal blood flow via increased splanchnic vasoconstriction. This attempt to maintain blood pressure, at the expense of the liver, kidneys, and other organs, appears to account for the high rate of hepatic and renal complications in exertional heatstroke.

Depending upon the intensity and duration of exercise hyperthermia, cardiovascular compensation begins to fail. At some point the individual ceases to work or collapses with one of the characteristic heat illnesses, e.g., heat syncope, heat exhaustion or heatstroke. This typically initiates diagnosis and therapy. Classic therapy addresses the basic physiological causes of distress and matches the treatment with the stress: 1. Cease the activity, 2. Lie the patient down, 3. Cool the body, and 4. Replace fluid-electrolyte deficits.

Heat Syncope

Even standing quietly in the heat before, or more commonly after, exercise results in an increase in venous volume in skin and dependent limbs which

increases the difficulty of maintaining adequate cardiac filling pressure. If filling pressure and stroke volume decline rapidly, a fall in cardiac output and blood pressure will occur and syncope will result. The diagnosis of heat syncope is based upon the observation of a short-term fainting episode in the absence of elevated rectal temperature. Recumbency, rest, avoidance of sudden or prolonged standing, and oral replacement of fluid-electrolyte deficits are adequate treatment. Heat syncope is typically categorized as a syndrome distinct from heat exhaustion (Table 1).

Heat Exhaustion: Volume and Fluid-Electrolyte Factors

Heat exhaustion is primarily a volume depletion problem. Dehydration due to salt depletion (either low dietary NaCl, high losses of NaCl, or both) results in a loss of extracellular fluid and a reduction of plasma volume, cardiac output, and blood pressure; it is neither characterized by thirst nor relieved by administration of salt-free fluids. Dehydration due to water deprivation or fluid losses is characterized by thirst and oliguria, and is completely relieved by the administration of water. These two forms of dehydration comprise the two widely-recognized categories of heat exhaustion: salt-depletion heat exhaustion and water depletion-heat exhaustion. Severe salt depletion-heat exhaustion produces a form of peripheral vascular collapse closely resembling traumatic shock; water depletion, with a comparable decline in extracellular fluid volume, does not produce peripheral vascular collapse and little or no protein leaves the vascular compartment (2). Water depletion is more likely to lead to heat stroke than salt depletion-heat exhaustion. Because of the similarities in symptoms, differentiation between these two forms of heat exhaustion may be very difficult. Hubbard et al. (3), in fact, emphasized that pure forms of salt depletion and water depletion heat exhaustion are rare, because body fluid

losses (e.g. diarrhea, vomitus, sweat, urine) involve mixed water and salt losses. Urine and plasma levels of sodium and chloride seem to be the only consistant diagnostic index.

Heat Cramps and Heat Exhaustion

Heat cramps are a frequent complication of heat exhaustion but sometimes occur with minimal systemic symptoms. Hyponatremia and hypochloremia may also be helpful in diagnosing heat cramps. Leithead and Gunn (4), for example, reported that healthy field laborers (n = 40) produced urine samples which contained 10g NaCl/L, while field laborers with heat cramps lost only 4g NaCl/L urine. Similarly, Talbot (5) observed serum sodium levels ranging from 121-140 mEq/L (normal = 135-145 mEq/L) in 32 heat cramp patients. We have previously described cases in which other syndromes (e.g. hyperventilation-induced tetany, gastro-intestinal infections) have been mistaken for heat cramps (6). Heat cramps in voluntary skeletal muscle affect a few muscle bundles only and as one bundle relaxes, an adjacent bundle contracts for 1-3 minutes. This gives the impression that the cramp wanders over the affected muscle. Although the pain of severe heat cramps is excruciating, the following treatment is rapid and effective: intravenous saline solutions (i.e. 0.5-1.0 L normal saline, a bolus of 10-20 ml of 23.5% hypertonic saline) or 1% oral NaCl solution (2 salt tablets crushed in 1 liter of water).

Volume Repletion

Oral rehydration therapy with appropriate fluids is generally prefered in ambulatory patients lacking symptoms of CNS dysfunction and free of vomiting and diarrhea. On the other hand, because exercise-induced heat exhaustion is primarily a volume depletion problem, intravenous therapy results in rapid recovery. Normal, young heat exhaustion victims of intense competition or

exertion may require up to 4 L of intravenous fluids. For example, the 1985 Boston Marathon was run on a warm day in April (75°F) at a time when few runners had acclimatized to heat (average daily maximum temperature in Boston during April, 1986 was 54.6°F). The medical tent at the finish line administered intravenous fluids to 158 cases of heat exhaustion. It was later estimated that 90% of these patients recovered and walked out of the medical treatment area within 15-20 min (7). Similar cases of rapid recovery from heat exhaustion have been reported by military medics, utilizing i.v. therapy, in Grenada and Germany. Furthermore, our review of the clinical literature indicates that there is no agreement on a preferred i.v. solution to utilize in the treatment of heat exhaustion. The fact that a glucose solution works as well as a saline solution underscores the fundamental role of volume depletion in heat exhaustion symptomatology.

Although a milder form of heat illness than heatstroke, heat exhaustion is the most common form of heat injury observed in athletic and military populations. The classical case of exercise-induced heat exhaustion with fluid-electrolyte depletion can be distinguished from heatstroke by: (a) the degree of central nervous system derangement, (b) rectal temperature below 40°C (104°F), and (c) non-significant changes in serum enzymes (e.g. LDH, CPK, ALT, AST) for 48 hours. However, there is a small subgroup of heat injury patients who have high rectal temperatures, hypotension, and confusion, who fall in the middle ground between heat exhaustion and heat stroke. When the diagnosis is in doubt, these patients should be treated for heat stroke, since this injury may be fatal.

Hyperthermia

Costrini (8) noted that the pathophysiologies of heat exhaustion and heatstroke may be very similar and hypothesized that they may represent a continuum of diseases rather than separate, distinct pathophysiologic entities. However, this must be considered in light of the fact that both heat exhaustion and heat stroke symptoms may occur independently of one another. Experimental studies on rats provide some support for Costrini's hypothesis. For example, we previously reported (9) that a temperature of 40.4°C represented a threshold hyperthermia, above which heatstroke mortalities occured in exercised heat-stressed rats. The mean (± SE) core temperature at exhaustion which produced a 50% mortality rate within 24h was 41.5 ± 0.1 °C. These results suggest a "continuum", in that the probability of mortality appeared directly related to the core temperature at collapse. These results also emphasize our observation that hypotensive heat injury victims may progress from severe heat exhaustion to heatstroke, if left unconscious and untreated.

In the forgoing animal model, the longer that exercise was maintained, the higher the core temperature rose and the more likely a serious case would occur. This is analogous to human victims of hyperthermia. These young, highly motivated, healthy individuals are essentially unaware of the seriousness of their hyperthermia, in contrast to the more familiar sensations of fatigue or exhaustion. For example, we have reported that an Olympic marathon runner produced metabolic heat in excess of 1,400 kcal/hr (10). Under conditions which limit heat dissipation, this rate of heat storage could have approximated 0.5°C min, and heatstroke levels of hyperthermia could have occurred within 10-12 minutes. The lack of pain and the apparent subtlety of symptoms of evolving hyperthermia often allow athletes, who are driven by pride and discipline, to voluntarily increase exercise intensity during the latter stages of competition.

Cooling Strategies

During the medical emergency of heatstroke, mortality is closely related to the duration and intensity of hyperthermia. But how effective is the process of cooling young heatstroke victims? Costrini et al (8) demonstrated that immersion of 13 exertional heatstroke patients (rectal temperature over 106°F) in a tub of ice and water, combined with massage of the extremities, resulted in a 100% survival rate. Although opponents of ice water immersion emphasize the negative aspects of intense cutaneous vasoconstriction in the cooling process, it must also be recognized that acute circulatory failure occurs in 80% of heatstroke deaths and that aggressive cooling markedly decreases cardiovascular complications in heatstroke victims (8,11).

In their 1959 review of methods of cooling hyperthermic subjects, Wyndham et. al. strongly denounced cooling with ice water (12). Yet, later labor statistics emanating from the same laboratory (13) indictated that the ratio of heatstroke fatalities to heat stroke incidents rose between 1969-1980, in spite of clear reductions in the incidence of heatstroke among miners. It is relevant that their treatment of heatstroke did not include ice water immersion. Peripheral vasoconstriction via cold water immersion represents a physiologically relevant means of restoring blood pressure. If induced prior to intravenous treatment, the risk of overzealous fluid administration, leading to pulmonary congestion or edema, may be avoided.

Since a delay in cooling heatstroke patients increases risk of serious injury, military medics attempt to cool the casualty in the field as close to the point of collapse as possible. The potential ice requirements for casualty cooling, combined with that necessary to provide cool drinking water, represents an overwhelming logistical burden, however. We calculate that the total ice

cost of cooling an 80 Kg man (from 45 to 37°C in a tub of water which has equilibrated with an air temperature of 43°F) to be 37.1 kg (82 lb) of ice. Therefore, the development and use of small, mobile water-chillers, to provide 15°C water for both drinking and casualty cooling, was a necessary compromise. In fact, Magazanik et al (14) reported that hyperthermic dogs were cooled as effectively in 15°C water as in ice water. If both ice and chilled water are unavailable, some degree of intravenous fluid replacement is indicated. Intravenous therapy offers the additional benefits of allowing other compounds to be added rapidly to the vascular compartment.

We have examined a promising adjunct to intravenous therapy, in collaboration with Dr. Andrew Young, Military Ergonomics Division, U.S. Army Research Institute of Environmental Medicine. This adjunct took advantage of the well-known cutaneous "flush" which is induced by nicotinic acid. We hypothesized that this "flush" would counteract vasoconstriction during cold water immersion, thereby promoting cooling of the body's core. Important to a complete understanding of these data is the fact that niacin's cutaneous flush occurs without altering blood pressure or heart rate. Our preliminary findings have been published (15), and indicate that 90 min oral nicotinic acid pretreatments resulted in lower minimal core temperature (35.2 - 36.6°C) than 90 min control water immersion trials (36.7 - 37.3°C). Additionally, body cooling was greater in 30°C water than in 25 or 28°C water, confirming that shivering and vasoconstriction counteract net heat loss. The striking feature of this finding was the magnitude of body cooling in relatively temperate water (30°C), which induced little shivering. The authors recognize that some optimal combination of nicotinic acid and intravenous Valium (to control shivering) may be used someday to induce rapid whole-body cooling in heatstroke victims immersed in even temperate water.

The Hypothetical Site of Heatstroke Pathophysiology: The Cell Membrane and the Sodium Pump

We have recently compiled a list (16) of hypothetical characteristics of a cellular site which would relate the physical effects of heat stress to the physiological manifestations of heat strain (Table 2). Cell membranes are in direct contact with hot blood, lymph and extracellular fluid, and must function in some way to balance the destabilizing effects of extracellular changes on the intracellular milieu. Basically, key extracellular factors increase the permeability of the cell to sodium ions/ and stimulate the sodium pump, which burdens the cell with an additional energy drain (the Energy Depletion Model).

Hyperthermia and exercise "drive" the sodium pump (16) in the following ways: (a) Hyperthermia increases intracellular acidity and this stimulates a Na^+-H^+ exchange. (b) Hyperthermia increases the kinetic energy and diffusion of ions in solution and this increases sodium permeability. (c) Heat storage is generally accompanied by hypohydration (sweating and voluntary dehydration) and hypohydration increases sodium permeability by simply increasing the extracellular sodium concentration. This suggests that the hyperthermia of dehydration is in part due to a general increase in cellular heat production. (d) Hyperthermia increases the neural stimulation frequency (nicotinic, cholinergic) necessary to maintain muscle force and this increases the sodium - potassium flux across nerves and stimulated muscles. (e) Hyperthermia and exercise produce regional ischemia (early splanchic, late cerebral) which increases regional acidosis and Na^+ flux. (f) Heat storage and exercise increase muscarinic and nicotinic cholinergic stimulation autonomically and voluntarily, which increases Na^+ flux.

Sodium potassium-ATPase activity is regarded (16) as the enzymatic equivalent of the sodium pump and accounts for a high proportion (20 to 45%) of the total energy used in resting cells. Active sodium transport is present in all cells. The active transport of either sodium or potassium requires the presence of the other on the opposite side of the membrane and takes place against both concentration and electrical gradients. This active transport is tightly coupled and requires that one molecule of ATP be hydrolyzed in order to return three sodium ions to the exterior of the cell, in exchange for two potassium ions. The velocity of active transport depends upon the concentrations of the cations at their respective binding sites and is remarkably sensitive to an excess of sodium ions inside the cell membrane. Because pumping activity increases approximately in proportion to the third power of the sodium concentration, a doubling of internal sodium concentration results in an eight-fold increase in ATP hydrolysis. Thus, any factor or condition which stimulates the flux of sodium into the cell will increase ATP utilization, will increase heat production, and will constitute an energy drain upon the organism.

The Energy Depletion Model

The Energy Depletion Model had its roots in two key observations: 1) There was an apparant paradox between the dogma that heat was the sole noxious agent precipitating heatstroke, versus the observation of a significantly higher morbidity and mortality associated with exercise-hyperthermia than equivalent hyperthermia alone (17). 2) Treadmill performance (work done at constant speed and grade in our non-sweating animal model) was significantly and inversely related to heating rate (16). In other words, the heat which the cell experiences (i.e. stored heat) adversely affects performance or increases

fatigue. Drug administration designed to worsen the situation (physostigmine or DNP) followed and extended the observed relationship (16).

This model suggested that an exercise-induced thermal imbalance led to an energy imbalance within the cells and required the hypothetical creation of an ion-pumping motor (the sodium pump) fueled primarily by glycolysis (6), and a set of conditions to run it at high speed to the point of overload and damage. The hypothetical accelerator was the rate at which sodium "leaked" into cells. This leak rate is now seen to have significant autogenerating or positive feedback potential, as shown in Figure 1. We have diagrammed this dynamic relationship between rate of heat gain, increased membrane permeability, cellular energy depletion and increased neurotransmitter activity as a viscious circle leading to reduced exercise-heat tolerance and significant morbidity and mortality.

The ultimate outcome of this proposed pathophysiological mechanism would depend on many factors, including the duration, intensity, and rate of heating, as well as variations in regional and local circulation. Without a doubt, the cascade begins with heat, is accelerated by acidity, and is progressivly worsened by hypohydration. This fits comfortably within the framework of our clinical experience and provides a clear rational basis for the importance and urgency of cooling, which reduces an additional source of heat production (ie. sodium - potassium ATPase activity). This is also consistent with the recognition by neurosurgeons and cardiovascular surgeons that hypothermia provides clinical protection from circulatory arrest by lowering the basal metabolic rate. Cooling restricts Na^+ channels, delays energy depletion, delays K^+ efflux, and stabilizes the cell membrane (18). This model, therefore, supports the essential nature of body cooling as a primary form of treatment in heatstroke.

References

1. Rowell LB. General principles of vascular control in: Human Circulation: Regulation during physical stress. New York, New York; Oxford University Press, 1986, pp 8-43

2. Elkinton JR, Danowski TS, Winkler AW. Hemodynamic changes in salt depletion and in dehydration. J Clin Invest 1946; 25:120-129

3. Hubbard RW, Armstrong LE, Evans PK, DeLuca JP. Long-term water and salt deficits: A Military Perspective. In: Predicting Decrements in Military Performance Due to Inadequate Nutrition. Washington DC: National Academy Press, 1986, pp 29-48

4. Leithead CS, Gunn ER. The aetiology of cane cutter's cramps in British Guiana. In: Environmental Physiology and Psychology in Arid Conditions. Leige, Belgium: UNESCO, 1964, pp 13-17

5. Talbot JH. Heat Cramps. In: Medicine. Baltimore: Williams and Wilkins Co, 1935, pp 323-376

6. Hubbard RW, Armstrong LE. The Heat Illnesses: Biochemical, Ultrastructural and Fluid-Electrolyte Considerations. In: Human Performance and Environmental Medicine, K.B. Pandolf, editor, Indianapolis: Benchmark Press, 1988, in press

7. Nash HL. Treating thermal injury: disagreement heats up. Physician Sports Medicine 1985; 13:134-144

8. Costrini AM, Pitt HA, Gustafson AB, Uddin DE. Cardiovascular and metabolic manifestations of heatstroke and severe heat exhaustion. Am J Med 1979; 66:296-302

9. Hubbard RW, Matthew WT, Linduska JD, Curtis FC, Bowers WD, Leav J, Mager M. The laboratory rat as a model for hyperthermic syndromes in humans. Am J Physiol 1976; 231:1119-1123

10. Armstrong LE, Hubbard RW, Jones BH, Daniels JT. Preparing Alberto Salazar for the Heat of the 1984 Olympic Marathon. Physician Sportsmedicine 1986; 14:73-81

11. O'Donnell TF, Clowes FHA. The circulatory abnormalities of heatstroke. New Eng J Med 1972; 287:734-737

12. Wyndham CH, Strydom NB, Cooke HM, Maritz JS, Morrison JF, Fleming PW, Ward JS. Methods of cooling subjects with hyperpyrexia. J Appl Physiol 1959; 14:771-776

13. Kielblock AJ, Schutte PC, Hitge JJ, Strydom NB. The recognition, treatment and diagnosis of heatstroke in the gold mining industry. Johannesburg: Chamber of Mines of South Africa, 1981, Research Report No. 33/81

14. Magazanik A, Epstein Y, Udassin R, Shapiro Y, Sohar E. Tap water, an efficient method for cooling heatstroke victims -- a model in dogs. Aviat Space Environ Med 1980; 51:864-867

15. Hubbard RW, Armstrong LE, Young AJ. Rapid hypothermia subsequent to oral nicotinic acid and immersion in warm (30°C) water. Am J Emerg Med 1988; in press.

16. Hubbard RW, Matthew CB, Durkot MJ, Francesconi RP. Novel approaches to the pathophysiology of heatstroke: The energy depletion model. Ann Emerg Med 1987; 16:1066-1075

17. Hubbard RW. Effects of exercise in the heat on predisposition to heatstroke. Med Sci Sports 1979; 11:66-71

18. Astrup J. Energy-requiring cell functions in the ischemic brain: Their critical supply and possible inhibition in protective therapy. J Neurosurg 1982; 56:482-497

Table 1 - International Statistical Classification of Heat Illnesses

992.0 Heat stroke and sunstroke: Heat apoplexy, heat pyrexia, ictus solaris, siriasis, thermoplegia

992.1 Heat syncope: Heat collapse

992.2 Heat cramps

992.3 Heat exhaustion, anhydrotic: Heat prostration due to water depletion. Excludes: salt depletion heat exhaustion (992.4).

992.4 Heat exhaustion due to salt depletion: Heat prostration due to salt (and water) depletion.

992.5 Heat exhaustion, unspecified: Heat prostration NOS.

992.6 Heat fatigue, transient

992.7 Heat edema

992.8 Other heat effects

992.9 Unspecified

Reprinted with permission from Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death. Geneva, World Health Organization, 1977, pp535

Table 2 - Site of Cellular Heatstroke Injury: Hypothetical Characteristics

* Common feature of all cells - especially nerves and muscles
* Temperature sensitive
* Related to cell volume changes
* Functionally related to endurance training
* Functionally related to the heat acclimatization response
* Functionally related to tolerance and fatigue
* Ability to generate heat
* Potential for inducing irreversible change

END

DATE

FILMED

6-1988

DTIC